TERRITORY HEALTH SERVICES
Centre for Disease Control

THE NORTHERN TERRITORY

# COMMUNICABLE DISEASES BULLETIN

NT

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*Congratulations to all the heroic service providers and volunteers who cleaned up after the Katherine and Douglas Daly River floods and kept things going throughout.*

## In the aftermath of the Katherine and Douglas Daly River floods

The recent floods in Katherine and the Douglas Daly River region gave CDC the opportunity to assess and review disease control priorities in disaster situations. While disasters and their public health consequences differ according to individual circumstances, a number of important lessons for disaster preparedness can be learned from the international and local literature on disasters (short list overleaf). The main dangers in the acute post-disaster phase are injuries and acute exacerbations of chronic diseases such as diabetes, especially if medical supplies run short.

Outbreaks of infectious disease after a disaster are only expected if there has been significant disruption of infrastructure, the environment and living conditions and diseases endemic to the affected area are more likely than exotic diseases. In a flood situation, outbreaks may result from ground water and food contamination (eg hepatitis A and diarrhoeal diseases) and vector control problems (eg Ross River and Barmah Forest viruses). Crowding in disaster shelters may facilitate disease transmission, particularly airborne infections and measles is the vaccine preventable disease most often associated with large outbreaks in refugee camps.

Katherine has not reported an increase in campylobacteriosis, salmonellosis, shigellosis or Ross River virus since the floods. In addition, there has been no apparent increase in the number of cases of hepatitis A (incubation period 15-50 days, usually 30 days) although it is still under enhanced

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surveillance. These statistics are a testimony to the prompt action taken by the Essential Services, Environmental Health, Medical Entomology and Katherine CDC staff and other service providers who re-established infrastructure and stressed the importance of personal and environmental hygiene (see Box).

Assessing the need for mass immunisation against hepatitis A and tetanus became a public issue during the recent floods. Post-disaster experience overseas and in Australia suggests that mass immunisation may not always be valuable in protecting against disease, and hence potentially diverts resources from priority activities. However, immunisation of high risk groups (eg relief workers entering the site) may be warranted, not least to prevent further burdening disrupted health care services. Therefore, hepatitis A vaccine and ADT were offered to all Essential Services staff who had direct contact with faecally contaminated water and food.

Basic hygiene and safety precautions will reduce the potential health impacts of disasters and CDC prepared the following fact list for residents of flood affected areas.

**HEALTH WARNING**

**Ensuring your family's health during and after the flood**

Take the following steps to prevent illness and injury:

**DO NOT SWIM IN FLOODWATER**

- Always wash your hands using soap before eating or preparing food.
- DO NOT drink river or bore water until it has been checked by THS or PAWA. Be aware of health warnings about the need to boil water or sterilise it with proper chemicals.
- Ensure that human and animal faeces is disposed of safely away from water sources (including disposable nappies).
- Don't eat food that may be spoiled.
- Report to the health centre if you develop diarrhoea or vomiting.
- Use mosquito repellent to avoid mosquito bites.
- Have a supply of your usual medication if you have a chronic illness, like diabetes or high blood pressure etc.
- Melioidosis disease occurs in the Top End - avoid contact with mud and muddy water by wearing protective boots, closed in shoes and heavy gloves when walking and working in these conditions.

**Take care when cleaning up. Avoid injury by:**

⇒ wearing protective boots, shoes and gloves

⇒ clean all cuts, sores and grazes immediately with water and soap or disinfectant and keep them covered

⇒ snakes may go into your home - avoid snake bites and seek immediate medical attention if you are bitten.

⇒ Make sure there are no dead animals within 200m of your home.

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# The effect of conjugate Hib vaccines on the incidence of invasive Hib disease in the NT

*Peter Markey, former District Medical Officer, Rural Services, Darwin*

## Background

CDC staff and clinicians Territory-wide are by now aware of the falling incidence of Hib disease in the NT since the introduction of the vaccine in 1993. Last year the manufacturers of the main vaccine used (PedvaxHIB®, Merck and Co), asked CDC to produce a report documenting this fall. This article is a summary of the report.

In the late 1980s, several reports in the literature documented a very high incidence of childhood infection caused by invasive *Haemophilus influenzae* type b (Hib) in the NT, particularly in Aboriginal infants under 12 months.[1,2] In 1993, both HibTITER® and PedvaxHIB® became available in Australia and were licensed for use in infants from 2 months of age. PedvaxHIB® was chosen for use in the NT, for both Aboriginal and non-Aboriginal infants, because of its demonstrated higher immune response after the first dose, its efficacy in the younger age group and its recommended regimen of three doses.

On 1 April 1993 the NT Childhood Vaccination Schedule recommended PedvaxHIB® to all children born after 1 December 1992. Several cases of invasive disease in older infants early in 1993 led to extra funding for the implementation of a "catch-up" program in July 1993, which included all children under 5 years of age (born after July 1988). Under this program, non-Aboriginal infants born before December 1992 were recommended HibTITER® (provided by the Commonwealth free of charge), and the Aboriginal infants of all ages were recommended PedvaxHIB®.

In late 1993, CDC launched a Hib vaccine promotion campaign specifically targeting the city of Darwin, as coverage rates in the Darwin Urban District had been below the high rates achieved elsewhere. This campaign, whose logo was "The Horrible Hib Monster", resulted in a significant increase in vaccine uptake in the targeted areas.[3]

## Methods

### Coverage rates

The data concerning Hib vaccine were extracted from the immunisation registers, merged, and converted to the SAS system, where they were analysed following the removal of duplicate records. In 1993, it was estimated that in the northern rural districts, 90% of children aged from 0-6 years living in participating communities were on the registers. The total number of children on the register in 1996 (21,500) was used as the denominator population for the calculation of coverage rates. Australian Bureau of Statistics (ABS) denominators were used in the calculation of vaccine efficacy. Analysis involved assessing, on a month by month basis, the proportion of children under five years who were immunised, so as to observe the uptake of the vaccine in the community following its introduction.

### Active case finding

From 1988 to 1994, there was no formalised data collection or notification of Hib disease in the NT. Earlier data (1985-88) were collected as part of a project by CDC staff and the incidence of the disease reported in the literature.[1] Until the mid 1990s, neither the hospital infection control units nor the laboratories had computerised records, and data were kept in an ad hoc fashion. Therefore, case-finding was done in a retrospective manner after discussing the problem with CDC, laboratory and hospital infection control staff in each location. As such, methods for case-finding varied from place to place.

From 22 December 1994, invasive Hib disease became a notifiable disease in the NT. The cases were entered on an EpiInfo database.

## Results

### Vaccine coverage

The increasing proportion of children under five years who had been immunised is illustrated in Figure 1. The proportion of children under 5 years of age who were considered adequately immunised increased throughout the study period to 75.2% at the end of 1996 (or 81.2% of those over 4.5 months). At that time there was a further 8.3 % who were considered partly immunised.

### Numbers of cases

There were 119 cases identified between 1989 and 1996, as illustrated by age-group and ethnicity in Table 1. The mean age of Aboriginal cases was 10.2 months (median 7.2 months) compared to 17.3 months (median 14.5 months) for non-Aboriginal cases. A large proportion (73.1%) of Aboriginal cases occurred below one year of age, compared with 42.3% of non-Aboriginal cases (Table 1).

**Table 1 Cases of invasive H. *influenzae* type b infection under five years, by age and indigenous status, NT 1989-96**

| | Aboriginal | | Non-Aboriginal | | Total | |
|---|---|---|---|---|---|---|
| | Number | % | Number | % | Number | % |
| **less than 4 mths** | 11 | 11.8 | 0 | 0.0 | **11** | **9.2** |
| **4-11 mths** | 57 | 61.3 | 11 | 42.3 | **68** | **57.1** |
| **12-23 mths** | 19 | 20.4 | 8 | 30.8 | **27** | **22.7** |
| **24-47 mths** | 6 | 6.5 | 7 | 26.9 | **13** | **10.9** |
| **48-59 mths** | 0 | 0.0 | 0 | 0.0 | **0** | **0.0** |
| **Total** | **93** | **100.0** | **26** | **100.0** | **119** | **100.0** |

**Table 2 Incidence of invasive Hib disease (cases per 100,000 child-years) by age-group and indigenous status, 1989-June 1993**

| Age | less than 4 mths | 4-11 mths | 12-23 mths | 24-47 mths | 48 mths or more | Total |
|---|---|---|---|---|---|---|
| **Aboriginal** | 447 | 1315 | 298 | 33 | 0 | **278** |
| **Non-Aboriginal** | 0 | 164 | 77 | 33 | 0 | **50** |
| **Total** | **177** | **621** | **164** | **33** | **0** | **141** |

## Incidence data

During the pre-vaccination era the incidence of invasive Hib disease in the Aboriginal population was over five times that in the non-Aboriginal population (Relative Risk 5.5; 95% CI 3.7-8.4; $p<0.001$). The age group four months to one year had the highest incidence in each group. The distribution is illustrated in Table 2. The decline in invasive Hib disease incidence compared with the uptake of the vaccine in the community is illustrated in Figure 1, where the three month moving average is plotted against the curve illustrating the proportion considered to be adequately immunised

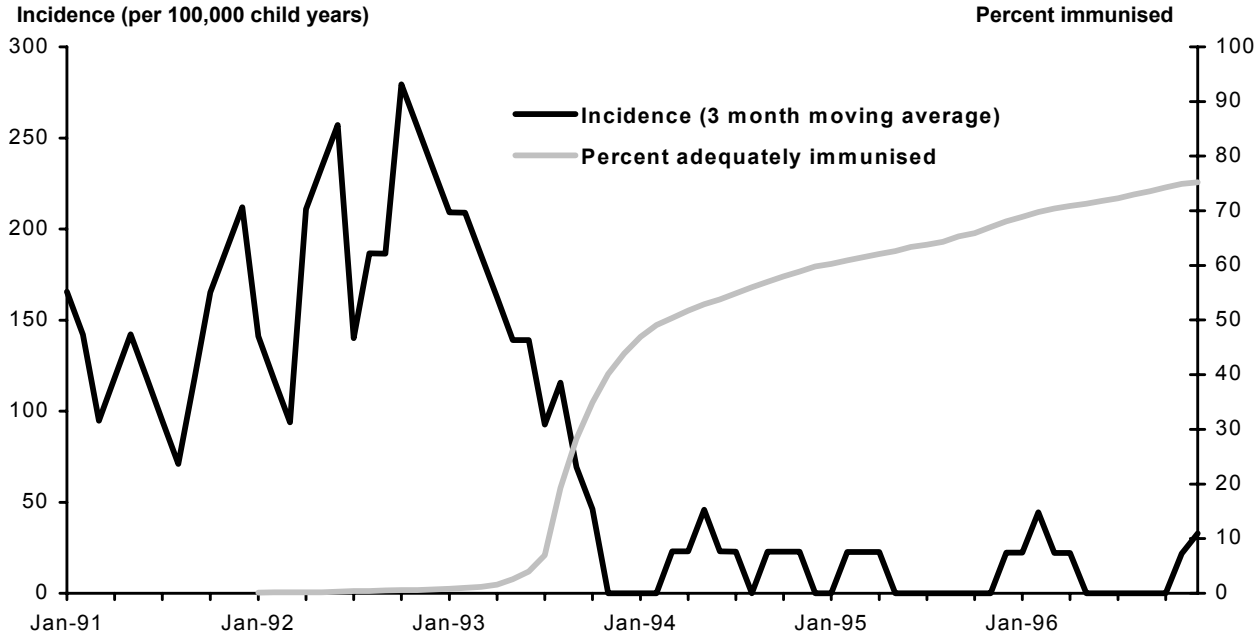


**Figure 1. Incidence of invasive Hib disease compared with overall Hib immunisation coverage, under 5s, NT 1991-1996**

**Table 3 Number of cases, incidence and relative risk of invasive Hib disease before and after the introduction of Hib vaccine, by indigenous status**

| | Pre-vaccination era | | Post-vaccination era | | | |
|---|---|---|---|---|---|---|
| | Cases | Incidence | Cases | Incidence | Relative risk | 95% CI |
| **Aboriginal** | 84 | 278 | 9 | 37 | **0.13** | **0.07-0.26** |
| **Non-Aboriginal** | 23 | 50 | 3 | 8 | **0.16** | **0.05-0.52** |
| **Total** | **107** | **141** | **12** | **19** | **0.14** | **0.08-0.25** |

**Table 4 Vaccine effectiveness results**

| Effectiveness | Pre-vaccination era | | Post-vaccination era | | | |
|---|---|---|---|---|---|---|
| | Cases | Child-years | Cases | Child-years | Effectiveness | 95% CI |
| **Overall effectiveness** | 107 | 75951 | 12 | 62293 | **86.3** | **75.2-92.5** |
| **Immunised with any Hib vaccine[(a)]** | 106 | 72786 | 5 | 41175 | **91.7** | **79.6-96.6** |
| **Adequately immunised with PedvaxHIB[(b)]** | 95 | 70254 | 1 | 29546 | **97.5** | **82.0-99.7** |

(a) At least one dose of any conjugate vaccine. The calculations are based on the population 2.5 months - 5 years.

(b) Two doses before 12 months of age or one dose after 12 months of age. The calculations are based on the population 4.5 months -5 years.

Notes.

1. Child-year calculations in the pre-vaccination era are derived from the estimated under 5 population for 1991.
2. Child-year calculations in the post-vaccination era are derived from the estimated under 5 population for 1995, and the immunisation coverage rates from the immunisation register.

## Vaccine effectiveness

The incidence of invasive Hib disease declined in the NT under five population from 141/100,000 during the pre-vaccination era to 19/100,000 in the post-vaccination era. The relative risk of invasive Hib disease after the introduction of the vaccine compared to before was 0.13 for Aboriginal children, 0.16 for non-Aboriginal children and 0.14 overall, with 95% confidence intervals (CI) given in Table 3. The incidence in the highest risk group (Aboriginal infants aged four to eleven months) fell from 1,315 to 185 per 100,000 child-years with a relative risk of 0.14 (95% CI: 0.06-0.33). The overall vaccine effectiveness was 86.3% while the effectiveness for that population which received at least one dose of any conjugate vaccine was 91.7% (Table 4). The effectiveness of PedvaxHIB® vaccine was 97.5% (95% CI: 82.0%-99.7%). There was only one case of Hib disease in a child considered to have been adequately immunised with PedvaxHIB®.

In Figure 2, the number of vaccine doses per month is plotted against individual cases per month. The effect of the "catch-up" program is evident.

## Cases after the introduction of the vaccine

There were 12 documented cases of invasive Hib disease after July 1st 1993. Six of the cases were infants who had received no vaccine and a seventh had received one vaccine only, 10 days prior to her illness. This seventh case became ill at aged 4.7 months and could have had the benefit of a second dose of PedvaxHIB® had immunisation taken place according to the schedule.

Three further cases were infants who had received one dose of vaccine and hence were partly immunised (two had received PedvaxHIB®and one HibTITER®). There were two cases who were adequately immunised; one of these had received one HibTITER® at age 21 months and the other a full course of PedvaxHIB®.

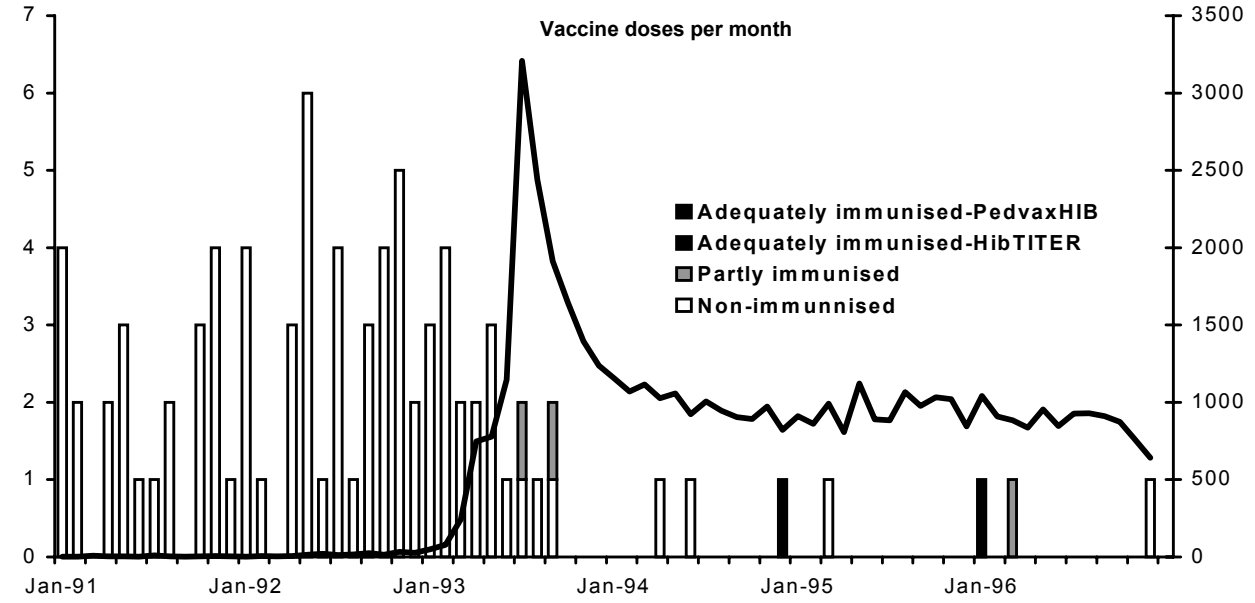


**Figure 2. Monthly cases of invasive Hib disease according to immunisation status, together with the number of Hib vaccines given, under 5s, NT 1991-96.**


## Summary

This report documents the decline of invasive Hib disease in the NT following the introduction of the conjugate vaccine in 1993. The incidence of invasive Hib disease fell to a seventh of its pre-vaccine level in both Aboriginal and non-Aboriginal infants and in the most at-risk age-group. There were two cases of documented vaccine failure, and ten other cases subsequent to the introduction of the vaccine.

The report illustrates both the success of the program and the effectiveness of the vaccine, while demonstrating the usefulness of the immunisation registers and the importance of timely immunisation.


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3. Mitchell D, Krause V. Evaluation of the NT's Hib promotion campaign. *NT Comm Dis Bull* 1994;2(4):4-7.


# Evidence associating measles viruses with Crohn's disease and autism inconclusive

The World Health Organization (WHO) recently reviewed the available literature and concluded there is insufficient biological, microbiological and epidemiologic evidence to support a hypothesised association between measles viruses and Crohn's disease, with studies either inconclusive or methodologically flawed.[1] WHO consequently recommends that measles immunisation should not be discontinued. Similarly, a recent case series report of 12 children suggested that MMR vaccine may trigger regressive development disorder (autism) with bowel disease.[2] This should be interpreted with caution as associations were temporal only and therefore cannot imply causality, particularly as millions of individuals have received measles vaccine since the 1960s with no previous reports of this syndrome.[3]

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3. Chen RT, DeStefano F. Vaccine adverse events: causal or coincidental? *Lancet* 1998;351:611-612.

# Update on HIV and Hepatitis C virus in the Northern Territory

*Jan Savage, NT AIDS/STD Program, Darwin*

## Human Immunodeficiency Virus

Since 1985 there have been 100 notifications of HIV infection in the Northern Territory (NT). The peak in notifications was in 1987, a little later than that experienced elsewhere in Australia. The number of cases reported annually has ranged from one in 1995 to 16 in 1987 (Figure 1).

Up until 1991, all HIV positive cases were found in non-Aboriginal people, however, since then twelve Aboriginal people have been diagnosed with HIV. In total, HIV appears less prevalent in the Aboriginal population, which comprises 27% of the NT population, but the trend of notifications in Aboriginal people is increasing. Elsewhere in the nation, there is little difference between the rates of HIV in indigenous and non-indigenous populations. The NT may unfortunately be approaching this situation.

**Figure 1 HIV notifications and indigenous status**

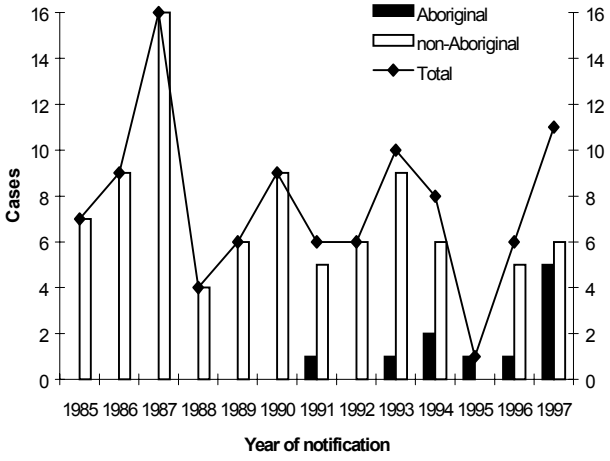



Table 1 shows the characteristics of HIV notifications in the NT compared to Australia overall from 1985 to 1997. Seven of the total of 100 HIV notifications (7%) have been in women, with more than half of these being reported in 1997. Of the 7, 2 have been Aboriginal (17% of all NT Aboriginal cases). This is similar to all indigenous women nationally accounting for 15% of all indigenous notifications and is in contrast to the overall national figure of 5% of HIV cases being in women.

Tables 2 and 3 refer to risk factors associated with HIV in the NT and Australia overall. Infection acquired through homosexual and or bisexual activity in men remains the most common route for transmission of the virus in the NT, accounting for 66% of all exposures (52% and 14% respectively). Heterosexual exposure is defined in 18% of all notifications and injecting drug use (IDU) is identified as a potential route of transmission for 12% of all cases, but is the exclusive method of exposure for only one case (1%). One child has been infected in utero and remained HIV positive. The mode of exposure is unknown or has not been recorded in ten cases (10%).

Almost all notifications originated from the Darwin region.

The number of HIV notifications over the period 1985-1997 is small, but as noted, there are differences from national rates and worrying trends which must be closely monitored to keep existing prevention programs informed. Heightened surveillance in at risk groups and groups where seroprevalence is largely unknown is recommended.

Notifications of AIDS cases (Figure 2) have remained fairly steady (between one and five cases per year). In recent years approximately half of these notifications have been in visitors to the Northern Territory.

**Figure 2 AIDS notifications, NT 1987-97**

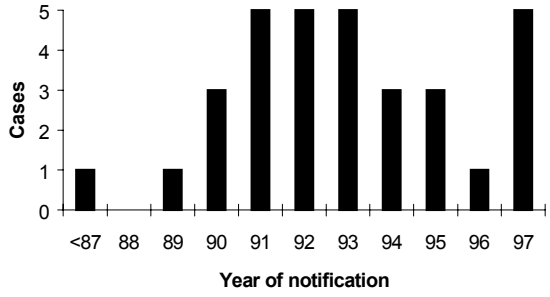



Figure 2 presents the epidemic curve of AIDS notifications for the NT. There have been no HIV related deaths in over twelve months which reflects the effects of advances in medical management that are now available.

Clinic 34 in Darwin and Alice Springs provides specialist clinical services for HIV positive people and consultation for health professionals caring for people with HIV/AIDS.

**Table 1 Characteristics of HIV notifications**

| | No of cases | Cumulative rate per 100,000 | Gender | | Indigenous status | | Cumulative NT rate per 100,000 | | Median age group at diagnosis |
|---|---|---|---|---|---|---|---|---|---|
| | | | M | F | Ab | non-Ab | Ab | non-Ab | |
| **NT 1985-Dec 97** | 100 | 56.3 | 93% | 7% | 12% | 88% | 24.6 | 68.6 | 35-39 years |
| **Australia 1985-Jan 97** | 16,700 | 91.2 | 95% | 5% | Equal rates reported for both | | | | 30-34 years |

**Table 2 Primary exposure categories for HIV notifications**

| | Homo/bisexual contact | IDU (only) | Heterosexual contact | Vertical transmission | Unknown | Other |
|---|---|---|---|---|---|---|
| **NT 1985-Dec 97** | 66% | 1% | 18% | 1% | 10% | 4%* |
| **Australia 1985-Jan 97** | 80% | 5% | 8% | 0.4% | 6.6% | |

*Includes infection through blood products, IDU not defined in other category

**Table 3 NT HIV trends 1995-97**

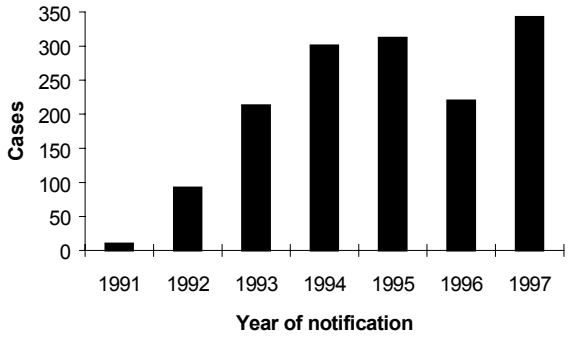
| | No of cases | Gender | | Indigenous status | | Exposure |
|---|---|---|---|---|---|---|
| | | M | F | Ab | non-Ab | |
| **1995** | 1 | 1 | 0 | 1 | 0 | one heterosexual |
| **1996** | 6 | 6 | 0 | 1 | 5 | one vertical<br>2 homo/bisexual<br>2 heterosexual<br>one unknown |
| **1997** | 11 | 7 | 4 | 5 | 6 | 6 homo/bisexual<br>3 heterosexual<br>2 unknown |

## Hepatitis C virus (HCV)

HCV is a laboratory based notifiable disease in the NT. Currently, unless information is volunteered by the clinician, no distinction is made between incident and prevalent infection. Duplicate notifications are screened and eliminated if an individual has previously tested positive in the NT, but not interstate.

HCV is the most commonly notified blood borne virus in the NT with injecting drug use (IDU) being the major route of transmission here and nationwide. It has been estimated that between 100,000 and 200,000 Australians have been infected with HCV, and among injecting drug users, there are between 8,000 and 10,000 new infections annually. Since testing began in 1991, there have been 1493 cases of HCV reported in the NT. In 1997 there were 343 cases notified. Figure 3 presents the epidemic curve for hepatitis C in the NT since testing began in 1991.

**Figure 3 Total NT notifications of HCV**




70.6% of cases (242 notifications) were male, 25.4% female (87 notifications) and sex was missing in 14 notifications. The ratio of male to

female is 3:1. Eighty seven percent of notifications were from the Darwin region. Approximately 66% of individuals with positive HCV serology were aged between 30 and 44 years and 85% were aged from 20 to 44 years.

In 1997, indigenous status was recorded as non-Aboriginal in 208 cases (60.6%), Aboriginal in 11 cases (3.2%) and was missing in 124 cases (36.2%). Given the large proportion of missing data for this variable, there is insufficient data to comment on risk factors for HCV in Aboriginal people.

There is presently no surveillance to assess incident cases, however the NT AIDS/STD Program will be requesting clinicians to provide more data on any patients with positive HCV serology in 1998.

In Darwin and Alice Springs, Clinic 34 provides clinical services for people with positive hepatitis C serology.

### Editorial

This review is a preview of the comprehensive analysis of all STD data conducted by the AIDS/STD Unit annually. Copies of the report will be available later in the year.

Surveillance to detect incident cases of hepatitis C stopped in 1995 after a trial period of 18 months. The NT detected about 3 incident cases annually. Details of the early surveillance system for acute hepatitis C and overall risk factor assessment appeared in:

Lush D. Improving our knowledge of hepatitis C. *NT Comm Dis Bull* March 1994;2(1):10.

Hepatitis C notification. *NT Comm Dis Bull* June 1994;2(2):9.

Current surveillance for HCV is exclusively laboratory based, hence the missing data on indigenous status.

## Non-Communicable Diseases Update: No.4. Message: Hypertension Control - choose your drug carefully, and monitor its effects

*Tarun Weeramanthri, Community Physician, CDC, Darwin*

The literature on hypertension is vast. The aim of this and following articles is to highlight recent changes in prescribing practice and overall management, and examine the reasons why there seems to be a discrepancy between prescribing practice and recommendations in authoritative hypertension guidelines. In particular this update will look at the use of the four major groups of antihypertensive medications: the 'older agents' namely thiazide diuretics (D) and beta-blockers (BB) and the 'newer agents' namely calcium channel blockers (CCB) and ACE inhibitors (ACEI). The use of other agents such as alpha blockers or the newer angiotensin receptor blockers will not be discussed in detail.

### Non-drug measures

Hypertension is present when blood pressure is persistently greater than 140 mmHg systolic and/or 90 mmHg diastolic. The further classification of hypertension by blood pressure level is contained in international guidelines.[1] The relationship between blood pressure and cardiovascular risk is continuous ie. the lower the blood pressure, the lower the risk of stroke and coronary events.[1] Modification of lifestyle factors (aiming for a moderate alcohol intake, no added salt diet, weight loss, decreased abdominal circumference and increased activity) can reduce blood pressure levels, and consequently lifestyle modification is recommended prior to drug treatment in most patients with mild hypertension.[2] Generally the effect of lifestyle modification is additive to that of medications, once medications are commenced.[3]

### Drug measures

The four groups of drugs (D, BB, CCB and ACEI) are equally effective in controlling blood pressure when used as first line agents.[4,5] All groups also favourably affect intermediate endpoints such as a reduction in left ventricular hypertrophy.[4] However, only D and BB have been shown to reduce adverse clinical events (such as stroke and cardiovascular events) in large trials. A recent meta-analysis

concluded that low-dose diuretics, but not high-dose diuretics nor BB, reduced the risk of overall mortality.[6] Overall, it is likely, but not certain, that the benefits of antihypertensive medications are due to lowering of the blood pressure, irrespective of the agent. Trials assessing the ability of CCB and ACEI to reduce adverse clinical events are currently being undertaken.

Quality of life considerations are particularly important in a condition, such as hypertension, which is usually asymptomatic. Quality of life deteriorates with diagnosis of hypertension ('labelling effect'),[7] and improves with treatment, whether using lifestyle modification or medications.[3,8] Data on quality of life differences between the four groups of drugs is limited, generally showing no significant differences, but with a few exceptions.[9]

The effects of the various classes of antihypertensive agents on lipid levels are demonstrable,[10] but they are dose dependent and not yet of proven clinical importance. In general, the following is true: high dose thiazides increase cholesterol and triglyceride levels, and can raise glucose levels; the effect of betablockers is variable; and CCB and ACEI are thought to have no adverse effects on metabolic parameters. Some researchers consider low dose thiazides to be metabolically neutral,[11] though a recent meta-analysis showed that thiazides adversely affect the lipid profile even at low dose.[12] Small changes in lipid levels for individuals may be important at a population level for the prevention of cardiovascular disease.

## Guideline recommendations

The fifth report of the US Joint National Committee (JNC) on Detection, Evaluation and Treatment of High Blood Pressure gave preferred status to D and BB as first line agents for the treatment of hypertension, unless there were specific contraindications, or there were special indications for other drugs.[13] (*The sixth report will be published in Archives of Internal Medicine later this year.)* The Australian Consensus Statement agrees in principle with this approach while noting that in a 'substantial proportion' of patients the selection of specific therapy is determined by the presence of coexisting conditions.[2] Clearly, while we still talk of 'first line' agents, a rigid 'stepped care' approach has been replaced by an approach tailored to the individual.

## Prescribing practices

In the absence of compelling cost effectiveness data, the only thing that can be said with any certainty is that the unit cost of CCB and ACEI is considerably greater than the older agents and that prescribers seem increasingly to favour the newer agents.[14] In the NT, in the last six months of 1995, 36% of the money spent on prescription items dispensed from the RDH pharmacy was spent on ACE inhibitors, representing 8.3% of the total pharmacy budget.[15] The cost of ACE inhibitors rose in each quarter from July 1994 through to December 1995, and is presumably still rising.

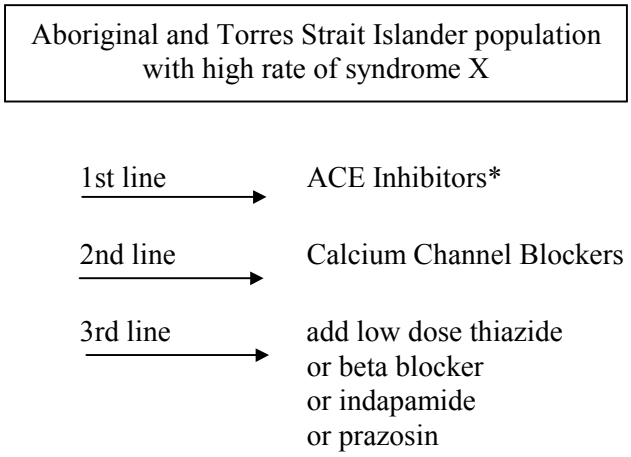
Prescribers either may be unaware of the randomised trial literature favouring D and BB in terms of clinical endpoints or they may be making a rational decision by choosing more expensive drugs because of their lesser potential for metabolic side effects, figuring that the goal of therapy should be to decrease overall cardiovascular risk, not just reduce blood pressure.[16] Effective drug promotion of the newer agents by pharmaceutical companies in a hugely profitable market must also be having some effect. It is of interest that CCB and ACEI are two of the four very commonly prescribed groups of drugs identified by the federal government for 'benchmark pricing' under Therapeutic Group Premium arrangements, operative from February 1, 1998.[17] This will result in savings to the government under the Pharmaceutical Benefits Scheme.

## Local factors

In the Aboriginal population, with its much higher rates of 'syndrome X' (insulin resistance, hyperinsulinaemia, central obesity, hypertension and dyslipidaemia),[18] and diabetes, albuminuria and renal failure in particular,[19] it may be more appropriate to be using the newer agents (especially ACEI) than in other practices elsewhere. ACEI may have a specific renal protective effect over and above that conferred by blood pressure lowering.[20,21] Indapamide is a newer non-thiazide diuretic, which also has vasodilating properties. It has no adverse metabolic effects and possibly has a specific renal protective effect.[22] However, there have been no large scale trials directly comparing its efficacy with standard treatments such as low dose thiazides.

In view of the above information, the Guidelines, Standards and Audit team operating as part of the NT Coordinated Care Trials has recommended in its

Hypertension Care Plan that ACEI should be first line treatment for hypertension in Aboriginal clients, with CCB as alternative agents. Low dose thiazides (or indapamide), beta blockers and prazosin (an alpha blocker) are listed as additional agents in clients whose blood pressure is difficult to control.




* ACEI are category D drugs in pregnancy, because they have caused fetal abnormalities when taken in the second and third trimesters of pregnancy. They should only be used in women of child bearing age if the woman is using a reliable contraception method, if she is counselled about the risks of the drug in pregnancy and the need to stop the drug if she thinks she may be pregnant or if she misses a period. CCB are considered generally safer in women of child bearing age, though they should also be stopped if pregnancy is confirmed.

## Critical question

The gap between authoritative guidelines that recommend D and BB as first line agents in hypertension and prescribers who increasingly opt for ACEI and CCB as first line agents, can best be explained by a difference in the importance assigned to clinical trial evidence (with its emphasis on definitive clinical endpoints) v's. the importance assigned to biological plausibility (with its emphasis on rational but unproven interventions and intermediate clinical endpoints).

The question prescribers should ask themselves when prescribing an agent for hypertension is as follows: *'Given that the older agents, namely D and BB, are the only medications that have ever been conclusively shown to favourably alter clinical outcomes in hypertension, are there particular reasons why I should choose a newer, more expensive, drug (CCB or ACEI) in this patient ?'*

## After prescription

Data from the 1988-1991 US National Health and Nutrition Examination Survey revealed that although the percentage of patients with hypertension taking medication was 73%, the percentage with a blood pressure below 140/90 on medication was only 21%.[7] So, regardless of the choice of first line medication, the prescriber has an even more important duty to monitor the response of the patient in terms of compliance, side effects, tolerability, and overall effect on quality of life as well as, of course, blood pressure control.

In the next issue of the *NT Communicable Diseases Bulletin*, we will look specifically at the treatment of hypertension in the elderly.

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# Death of a five year old from meningococcal disease in Darwin
# A case of unprecedented public alarm

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On Saturday, 25 October 1997 a five year old boy died in the Intensive Care Unit (ICU) of Royal Darwin Hospital (RDH) from meningococcal disease. While disease is expected throughout Australia during the late winter and early spring months, and deaths occur, this case was remarkable in the Northern Territory with respect to the unprecedented public response to media reports of the death.

## Case History

The child became unwell on the evening of Friday, 24 October with the non-specific symptoms of fever and a headache and had no prior illnesses that week. There was no preceding history of travel outside the Darwin area. He was seen by a private medical practice that evening and returned home after examination. The following morning he presented to RDH with fever, headache, confusion and a widespread purpuric rash and was admitted to ICU with a provisional diagnosis of meningococcal disease. By mid afternoon Saturday his condition deteriorated, and he died in the early evening. *Neisseria meningiditis* was cultured from the first set of blood cultures taken on arrival at RDH. In addition, organisms resembling N. *meningiditis* were seen on agar cultures of aspirate from a purpuric lesion on the child's back the next morning. The isolates were sent to the National Neisseria Network and identified as Group C, Type 2aP1.5.

## Public Health Response

The public health response to this case began on the Saturday afternoon and initially involved tracing the immediate family contacts and intensive care staff and providing them with prophylaxis. On Sunday morning, the Principal of the child's school was contacted and phone numbers for his immediate classmates obtained to organise prophylaxis and tell them of information sessions to be held at the school on Monday. By Sunday evening, all but two parents had been contacted.

Throughout the following week the public health action taken included:

- continuing contact tracing and the administration of either prophylactic oral

rifampicin or intramuscular ceftriaxone to close contacts which, in this case, were identified as household contacts, relatives who had kissed and cuddled the child and members of the child's morning preschool group;

- information sessions for staff and parents at the school on the Monday morning;
- onsite Centre for Disease Control (CDC) staff at the school and Palmerston Community Care for two days to give advice, reassurance and information to concerned parents and the general public;
- media releases, a press conference and interviews;
- contacting by phone and fax all listed general practitioners in the Top End to inform them of CDCs response to the case and enhance surveillance for further cases;
- the distribution of fact sheets to Community Care Centres, general practitioners, RDH doctors, child care centres and school children; and,
- responding to public calls by CDC, RDH and other THS departments.

By Monday afternoon, all close contacts considered most at risk (ie household contacts and immediate classmates) had been given prophylaxis. By Thursday, 30 October, a total of 143 contacts had been identified and 132 had been given preventive antibiotics. Of the remaining 11, six declined as they felt treatment was not necessary, three did not present (or respond to follow-up calls) after medications were arranged for them and two could not be contacted despite leaving messages at the school.

## The media

The media were aware at 11am on Sunday morning (how is unclear) and a television interview was held shortly after lunch. The news broke as a leading story by the sports newsreader on the 6pm Channel 8 news and was on the front page of the NT News on Monday morning under the headline "Darwin boy, 5, victim of killer bug".

The case generated a media frenzy in Darwin for three days, involving interviews with CDC staff, the Director of Microbiology, the Minister for Health, the School Principal, the child's mother and parents of children at the preschool. There was local and national television, radio and newspaper coverage. A televised interview with the child's grieving mother was followed by a ministerial response and a coronial investigation. Important facts about meningococcal disease from the press conference held at CDC were not conveyed to the public, and inaccurate visual and vocal reports that vaccines were being administered occurred.

On Monday morning, the government switchboard was jammed and the RDH switchboard, Emergency Department triage, CDC, and other health service providers in the Darwin region were overwhelmed with public calls. The major foci of inquiries were the level of risk to children, how to recognise disease and why vaccinations and antibiotics were not being offered to all Darwin children.

## Lessons learned

This case was unique in Darwin with respect to both media coverage and the public reaction it generated. Previous meningococcal cases in the NT this year had passed virtually unnoticed by the Darwin public, despite reports in the NT newspapers.[1,2] It is likely the response to this case resulted from the rapid death of the child, recent national coverage of the disease, interstate deaths and vaccination campaigns in Sydney. The level of public alarm was disproportionate to the degree of risk and probably precipitated by the public's feelings of lack of control and lack of information about the disease. The experience has highlighted the importance of a coordinated, uniform response to cases and public concern that is concise, evidence based and easily understood by the general public.[3]

Three essential messages need to be made clear to both the general public and health service providers:

1. assure the public that everything possible is being done and that nationally endorsed, best practice guidelines for controlling meningococcal disease are being followed;
2. inform them early of the appropriate response to a single case, which is contacting tracing, preventive treatment with appropriate antibiotics (which are sometimes given intramuscularly) and early recognition and intervention should symptoms arise. Importantly, ensure that local general practitioners are notified early and that they have access to meningococcal control guidelines;
3. clearly state who is considered a contact, why antibiotics are the appropriate response and emphasise that vaccinations are only indicated in the event of a community outbreak where there is

evidence of ongoing transmission in a defined, at risk population and an appropriate vaccine is available.

It is essential that health professionals and the public are aware of the limitations of the current vaccine, which include:

a) the poor immunogenicity of the serogroup C component of the vaccine in young children, especially those under 4 years of age;
b) the lack of protection against all Group B disease that accounts for approximately 60% of Australian cases;
c) the short duration of antibody response in children and adults;
d) its failure to reduce carriage rates, and
e) the 10-14 day delay between vaccination and the production of adequate levels of protective antibodies, and therefore plays no role in immediate protection from disease.[4]

The method and timeliness of communicating the facts is an issue which was addressed during a debriefing exercise. On the Monday morning, CDC, the RDH switch and other health service providers were overwhelmed with telephone calls and repeated, sensational media headlines made it difficult for the public to absorb the facts. In this case, timely communication was hindered by its occurrence on a weekend, with the media reporting the story within 24 hours of the death, before official press releases were issued and while appropriate public health action was being carried out (eg. contacting those immediate contacts at risk). Strategies considered for the future management of similar situations include establishing a 1800 telephone hotline and purchasing a full page advertisement in the NT News to communicate the facts. This experience also underscores the importance of general practitioners and other health professionals having ready access to, and being familiar with, current best practice control guidelines throughout the year so they are prepared for cases as they occur and the public attention they may generate.

## Acknowledgements

Disease Control would like to extend their thanks and appreciation to all involved in the response to this case. In particular, we would like to thank the staff of the Royal Darwin Hospital switchboard, Emergency Department and Pharmacy; the RDH Doctors who both notified CDC early and initiated contact tracing; the team at Media Liaison; the staff at Palmerston Community Care Centre; and Sue Jose (Principal), teachers, parents and children of Moulden Park Primary School.

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# Lessons from a case of meningococcal eye disease

*Sandra Thompson and Alyson Alway, CDC, Alice Springs*

In early February 1998, a 7 month old baby from a remote community was admitted to Alice Springs Hospital following two weeks of diarrhoea and one day of vomiting. On admission the infant was dehydrated with sunken eyes, reduced skin turgor and a dry tongue, lethargic, sleeping when left alone and irritable when disturbed. Weight recorded 17 days before the onset of the diarrhoea showed that the infant had since lost 930g. In the admitting notes it was recorded that the child had a temperature of 38.7°C, cervical nodes and sticky eyes bilaterally. The admitting diagnosis as recorded was diarrhoea (known to be due to cryptosporidia), vomiting and failure to thrive. After a septic work up which included blood cultures, lumbar puncture, eye swab and faeces microbiology, the child was treated as pneumonia and commenced on intravenous penicillin.

Two days after admission the results of the eye swabs were available. Four organisms had been grown: *Neisseria meningitis* +, *Streptococcus pneumonia* ++, *Branhamella catarrhalis* ++ and *Staphyloccus aureus* ++. The result was phoned through to Disease Control. The lumbar puncture

and blood cultures were negative. The infant was moved to a single room and IV penicillin continued.

NHMRC booklet *Guidelines for the control of meningococcal disease in Australia*.[1] They state that despite secondary cases to meningococcal eye cases being rare, that they should be managed like invasive meningococcal disease. All close contacts of a patient with meningococcal disease should be offered antibiotic chemoprophylaxis; this includes all household contacts and those who have spent more than 24 hours with the index case in the week preceding the onset of illness. Prophylaxis is undertaken to eliminate nasopharyngeal carriage of the organism from asymptomatic contacts and to prevent subsequent transmission and secondary invasive disease.

The mother received rifampicin prophylaxis and the infant received a stat dose of ceftriaxone at the conclusion of five days of intravenous penicillin. Ward room contacts were also treated with rifampicin. Disease Control in Alice Springs attempted to contact the clinic in the remote community to advise about the need to treat contacts with rifampicin. For two days the clinic phone was unanswered; when contact was made there was no rifampicin syrup available in the clinic with which to treat child contacts. With the tyranny of distance, in this instance household contacts were not treated.

What public health management is recommended for meningococcal eye disease? We consulted the

For optimal management of future meningococcal contacts in similar circumstances we suggest the following points be considered:

- Clinics have a telephone answering machine to be used when no one is in attendance.
- Disease Control should send a facsimile to the clinic where the phone is unanswered indicating the urgent need to make contact with Disease Control.
- Disease Control notify the DMO for that community about what action is needed.
- Disease Control maintain a list of alternative contact numbers to be used in emergencies. (In this instance, the nurse was actually in Alice Springs on one of the days and had a mobile phone with her. We also now have the clinic sister's home phone number.)
- Ceftriaxone IM be used for prophylaxis rather than rifampicin if rifampicin is unavailable.

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# Surveillance of meningococcal disease in the NT

*Angela Merianos, CDC Darwin*

## Introduction

Invasive meningococcal disease is an uncommon disease, but one of great public health importance because of its rapid clinical evolution, especially the very high risk of secondary cases among untreated household contacts and a case fatality rate of 5-15%. CDC makes every attempt to ensure that it is notified of all cases and that the quality of data is high. These characteristics make it a good indicator disease for the evaluation of a notifiable disease surveillance system; if the completeness of notification and data quality are poor for meningococcal disease, then we expect an even greater level of under reporting with other, more common, conditions.

A case of meningococcal disease requires urgent public health action to aid in the prevention of secondary cases. It is one of a small number of communicable diseases that is marked for:

1. immediate doctor and laboratory notification by phone, fax (or email) in all Australian jurisdictions for confirmed and probable cases; and
2. contact tracing and the administration of prophylactic antibiotics, in the form of rifampicin or ciprofloxacin orally or ceftriaxone by intramuscular injection, to be completed ideally within 72 hours of notification of the index case. The usual incubation period for meningococcal disease is 3-4 days, so that prophylaxis should be administered within this time frame to prevent secondary cases.[1]

## Methods

The completeness of data held by CDC on invasive meningococcal disease was determined by a retrospective audit of three data sources:

1. NT wide notification data from January 1992 to December 1997 held in annual computerised datasets at the Centre for Disease Control (CDC) in Darwin;
2. NT wide hospital separation data (HSD) provided by the Epidemiology and Statistics Branch, Territory Health Services; and
3. laboratory data kept as either computerised records and/or a log book of bacterial isolates grown from sterile sites (mainly cerebrospinal fluid and blood). These data were supplied by Royal Darwin, Alice Springs and Tennant Creek Hospitals. Katherine District Hospital laboratory does not keep individual records of isolates and was unable to assist with this evaluation. RDH receives isolates from both Katherine and East Arnhem.

*The NT Communicable Disease Surveillance System (NTCDSS)*

Disease Control units are located in each regional centre of the NT. They are responsible for local disease surveillance as part of the NT Communicable Disease Surveillance System, and for disease control activities. They transmit notification data to CDC, Darwin, fortnightly for transmission to the National Centre for Disease Control, Canberra. All State health authorities participate in the National Communicable Diseases Surveillance System, a core dataset of communicable diseases that are notifiable in all States and Territories. At the time of writing the national consensus was that a notifiable disease is reported by the jurisdiction that first diagnoses and treats the case irrespective of the patient's State of usual residence. This has since changed.

*Hospital separation data*

The hospital separation dataset includes the discharge diagnosis of inpatients of all NT public hospitals. The primary diagnosis and co morbidities are coded using the Australian Version of the International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM) codes.[2] ICD-9 categories 036.0-036.9 were used to identify cases of meningococcal infection. These categories include the following meningococcal infections: meningitis, encephalitis, meningococcaemia, meningococcal adrenal syndrome, carditis, optic neuritis, arthropathy, and meningococcal infections, unspecified. The hospital separation data provided to CDC included a non HRN unique identifier so that patients admitted to more than one hospital or readmitted for the same illness episode can be identified. These patients were only counted once in the tally of meningococcal disease cases. Cases appearing on each dataset were then matched by date of birth or age, sex and district of isolate origin.

**Table 2 Concordance between hospital separation data and the NT Communicable Diseases Surveillance system by district of report, January 1992 - December 1997**

| District | Both HSD & NTCDSS | HSD only | NTCDSS only | Total | Concordance (%) |
|---|---|---|---|---|---|
| Alice Springs | 11 | 3 | 2 | 16 | **69** |
| Barkly | 1 | 0 | 0 | 1 | **100** |
| Darwin | 17 | 4 | 3 | 24 | **71** |

## Results

Table 1 presents the total number of cases of meningococcal disease in the period of observation and indicates in which dataset cases appeared. A total of 59 cases of meningococcal disease occurred from January 1992 to December 1997; 51 cases appeared in the hospital separation data and 49 cases were reported to CDC, Darwin. Concordance between the two data sources was 69% overall. Concordance improved over time from 44% in 1992 to 87% in 1997.

**Table 1 Concordance between hospital separation data and the NT Communicable Diseases Surveillance system by year of diagnosis, January 1992 - December 1997**

| Year of diagnosis | Both HSD* & NTCDSS† | HSD only | NTCDSS only | Total | Concordance (%) |
|---|---|---|---|---|---|
| 1992 | 7 | 6 | 3 | 16 | **44** |
| 1993 | 2 | 1 | 0 | 3 | **67** |
| 1994 | 4 | 0 | 1 | 5 | **80** |
| 1995 | 6 | 0 | 2 | 8 | **75** |
| 1996 | 9 | 3 | 0 | 12 | **75** |
| 1997 | 13 | 0 | 2 | 15 | **87** |
| **Total** | **41** | **10** | **8** | **59** | **69** |

* Hospital separation data

† Northern Territory Communicable Diseases Surveillance System data

Table 2 shows concordance by NT district for the 59 cases among NT residents. Excluding the Barkly where only one case was admitted to hospital and reported to CDC, concordance ranged from 60-71%.

| | | | | | |
|---|---|---|---|---|---|
| East Arnhem | 9 | 2 | 2 | 13 | **69** |
| Katherine | 3 | 1 | 1 | 5 | **69** |

This review also detected missing data in the NTCDSS. These included missing dates of birth (4 cases), details of indigenous status (3 cases) and an error in the recorded indigenous status. In addition, there was one case that appeared on both datasets (established by concordance in sex, indigenous status and dates of admission to hospital and reporting to CDC), but with a different date of birth. Most of the missing data occurred in 1992-94.

Table 3 presents the data on meningococcal isolates from sterile sites provided by the Royal Darwin and Alice Springs Hospital laboratories.

**Table 3 Serogroups of sterile site meningococcal isolates cultured at RDH and ASH laboratories. Comparison with serogroups recorded on the NTCDSS**

| District | | Serogroup | | | | | On NTCDCSS | |
|---|---|---|---|---|---|---|---|---|
| | Year | B | C | W135 | U/K | Total | B | C |
| Darwin | 94 | 0 | 3 | 0 | 0 | 3 | 0 | 2 |
| | 95 | 2 | 2 | 1 | 0 | 5 | 2 | 1 |
| | 96 | 4 | 4 | 0 | 0 | 8 | 3 | 3 |
| | 97 | 2 | 1 | 0 | 4 | 7 | 1 | 1 |
| Darwin Total | | 8 | 10 | 1 | 4 | 23 | 6 | 7 |
| Alice Springs | 92 | 1 | 4 | 0 | 0 | 5 | 1 | 4 |
| | 94 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | 95 | 0 | 1 | 0 | 0 | 1 | 0 | 1 |
| | 97 | 4 | 2 | 0 | 0 | 6 | 4 | 2 |
| Alice Springs Total | | 5 | 7 | 0 | 0 | 12 | 5 | 7 |

RDH provided records from 1994 onwards. There were 23 sterile site isolates cultured at RDH and 12 from ASH. The serogroup result was available for all isolates cultured at ASH and all were reported to the NTCDSS. 42% of Alice Springs isolates were serogroup B meningococci and the remaining 58% were serogroup C. 42% of the 19 RDH isolates for which a serogroup result was available were serogroup B, 53% were serogroup C and 5% were W135 (one isolate).

the dataset although included as a question on the *Doctor/Hospital Notification of Infectious Disease* form. This means that some of the cases of meningococcal disease may have been notified in error ie were not sterile site isolates. This may include isolates from eyes and pharynx.

The serogroup result was available for 100% of cases notified to CDC in Alice Springs. Determining the concordance between the RDH isolates and NTCDSS data for the Darwin district is more difficult; only some results could be linked to specific patients on the NTCDSS and the origin of the isolate by district could not be determined. Therefore, it may be that some of the isolates were cultured from patients diagnosed in East Arnhem or Katherine districts.

## Discussion

In terms of absolute numbers, the NT diagnoses a small number of cases of meningococcal infection each year. This review shows that the quality and completeness of reporting cases of meningococcal disease to CDC has improved over time in all NT districts but still has room for improvement. This has been achieved through continuing education of hospital based medical staff about the importance of early notification for public health action, a high level of cooperation between laboratory staff and CDC, and a greater awareness by CDC staff about the need for a complete core dataset.

### Limitations of the current meningococcal disease surveillance system

There are several limitations of the current data held by CDC:

- Failure to consistently record the date that the local Disease Control unit was notified of a case of meningococcal disease as a data item of the NTCDSS core dataset. There is provision for recording the notification date on the NT *Doctor/Hospital Notification of Infectious Disease* form and should be filled in by all Disease Control staff when accepting phone notifications. Failure to record this date means that we are unable at present to determine the delay between diagnosis and notification.
- Fields on the *Doctor/Hospital Notification of Infectious Disease* form are inconsistent with fields in the database. This means that the onus is on the surveillance officer following up a case to collect the additional information.
- The site of the isolate is rarely indicated on

These problems will be addressed in 1998.

We are unable to adequately explain the cases that appeared on the NTCDSS from 1992-96 but not on the hospital separation dataset. Some cases presented as outpatients with a febrile illness and subsequently diagnosed with meningococcal

bacteraemia when blood cultures, taken as part of the septic workup, grew *Neisseria meningitidis*. Until recently names and/or HRNs were removed from regional data before being sent to CDC Darwin so that our staff were not able to establish whether the patient was admitted for treatment. Some of the cases may have been notified on the basis of clinical criteria (probable cases). No ICD-9 search was conducted under category 320.9, meningitis due to unspecified bacteria (bacterial not otherwise specified (NOS), purulent NOS, pyogenic NOS, suppurative NOS). Some may have been isolates from non sterile sites. The discrepancy between case numbers in 1997 is explained by the up to three month delay before hospital separation data are coded and computerised.

The recent community and media scare following the death of a five year old boy from meningococcal septicaemia in Darwin is a strong reminder of the importance of early notification, accurate information and a coordinated approach between clinicians, the laboratory and public health. Cases should be reported as soon as a patient is assessed as a probable case, and definitely within 12 hours of diagnosis. Household contacts have up to 1000 times the risk of meningococcal disease in the first week after diagnosis in the index case than the normal population.[3] Contact tracing for the administration of prophylaxis within the prescribed 72 hours aims to eliminate nasopharyngeal carriage of *N meningitidis* in asymptomatic contacts, thereby preventing transmission and secondary invasive disease in further contacts.

The following recommendations have arisen from this evaluation:

*Regarding CDC:*

i. Adoption of the national reporting form for meningococcal disease developed by the NHMRC Working Party on Meningococcal Disease.[1] This form is used to collect demographic, risk factor, pathology and outcome data in a systematic way. The onus lies on surveillance officers rather than data entry officers to ensure that data are complete. This was agreed upon at the recent meeting of surveillance officers held in Darwin 12 February 1998.

ii. Ensuring that data quality checks occur regularly so that missing data can be completed. This is especially important for details that describe the occurrence of disease in time, place and person.

iii. Transmission of unique identifier details to CDC Darwin for NT wide surveillance system evaluation (family name, given names and/or HRN).

*Regarding laboratories:*

Recording data using a system that enables rapid case identification eg HRN, and core demographic details (DOB, sex, district of isolate origin) as well as the serogroup, serotype, serosubtype and antibiotic sensitivity.

*Regarding hospital separation statistics:*

i. Ensuring that coding is accurate and co morbidities recorded appropriately.

ii. Ensuring the inclusion of the HRN rather than a computer generated number so that patients can be matched to cases reported to the NTCDSS. Discrepant findings can then be investigated. This has been successfully negotiated with the Epidemiology and Statistics Branch.

## Acknowledgements

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# To BAD or not to BAD?

## Or do bacterial antigen detection (BAD) kits in clinical use on CSF specimens offer any advantage over routine CSF Gram stain and culture?

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## Introduction

Latex agglutination of capsular polysaccharide bacterial antigens has been performed on CSF specimens at Royal Darwin Hospital (RDH) if the white cell count of the sample is greater than or equal to 30 cells/microlitre, and occasionally at the expressed request of the treating doctor. In theory, with antibiotic pretreated patients, it was hoped that the tests would improve the numbers and speed of detection of bacterial meningitis.

Anecdotally, however, in our institution the clinical performance of the test has been variable during some years of practical experience with BAD kits. Recent results from other centres are conflicting. There appears to be a difference in findings between developed and less developed countries, with the latter demonstrating an increase in detection of bacterial meningitis through the use of BAD testing.[1,2] Developed countries have failed to demonstrate the same findings,[3] with one study querying whether the fall of classic pathogen rates made the test less useful.[4] In the NT we have sophisticated testing facilities but high rates of disease, so it was important to look at our own data. We therefore performed a retrospective review of all bacterial antigen detection tests on CSF specimens over the last two years in order to gain a better understanding of their value in the laboratory of RDH, looking at cost, accuracy of detection and turnaround time of diagnostic kit use.

## Methods

All records of CSF specimens were assessed over a nineteen month period, 1 January 1995 to 31 July 1997 and results recorded in a database. Bacterial antigen detection test performance in other clinical specimens (eg urine) was not assessed in the study as it was not always possible to differentiate between colonisation and infection. All patient charts were reviewed from specimens that were positive on culture, Gram stain or BAD test. The positive cultures were cross-referenced with a logbook of all significant cultures kept in the microbiology laboratory. Criteria for the diagnosis of bacterial meningitis were two of three of the following: a positive CSF culture, Gram stain or a highly suggestive clinical picture. The clinical picture included CSF parameters (low glucose and high protein), a high neutrophil count and a compatible clinical illness which responded to appropriate antibiotics.

Sensitivity = 28.6%
Positive predictive value (PPV) = 85.7%
Specificity = 98.7%
Negative predictive value (NPV) = 83.1%

If the 4 cases of bacterial meningitis for which no BAD test is available were removed from the equation, the sensitivity is still only 33.3% and the NPV would be 86.1%. It is important to remember however that other organisms cause meningitis, and the battery of BAD tests includes only *N. meningitidis* serogroup B or A/C/Y, *S. pneumoniae,* Group B Streptococcus, *E. coli* K1 and *H. influenzae* type b antigens.

## Results

A total of 557 CSF specimens were collected over this period. Of these, 96 had BAD testing performed (Table)

**Table Number of bacterial meningitis cases detected using BAD test**

| Bacteria | Bacterial meningitis* | Detected by BAD test |
|---|---|---|
| *N. meningitidis* | 7 | 3 |
| *S. pneumoniae* | 8 | 3 |
| *E. coli* | 2 | 0 |
| Other | 4 | N/A |

* Detection of bacterial meningitis = 2 of 3 of positive smear or culture, or clinical picture.

There were no cases of *H. influenzae* or Group B streptococcal meningitis over the period studied. Of the *N. meningitidis* specimens, four were serotype B, two were serotype C and one was seen on Gram stain but failed to grow on culture. The BAD test detected two of the cases of serotype B and one of the serotype C cases.

The four 'other' specimens were from patients with culture proven bacterial meningitis for which no BAD test kit was available. Two cultured *Listeria monocytogenes*, one *Burkholderia pseudomallei* and there was one mixed infection with *Staphylococcus epidermis* and streptococci from a patient with a shunt in situ.

The sensitivity and specificity results for the 96 specimens are set out in the table below.

| | Bacterial meningitis* | |
|---|---|---|
| Bacterial antigen detection | Present | Absent |
| Positive | 6 | 1 |
| Negative | 15 | 74 |

* Detection of bacterial meningitis = 2 of 3 of positive smear or culture, or clinical picture.

The battery of bacterial antigen detection kits costs around $15 per CSF specimen. If we performed BAD testing on all CSF specimens, the cost per positive test would be $1392.50. Even though the laboratory does not routinely perform tests on CSF specimens with less than 30 cells/μL, there were 32 specimens that were BAD kit tested at the treating doctor's request. None of these were positive on BAD, Gram stain or culture. Even when only specimens with cell counts over 30/μL (and the above at physician request) were tested, the cost per positive result was still $240, remembering that this test did not pick up all cases.

Most important of all, there were no instances of culture confirmed bacterial meningitis where Gram stain was negative and BAD testing was positive. In other words, all BAD true positives were confirmed by positive Gram stain, so they did not speed up the diagnosis for clinicians treating the patient, or pick up any cases that the Gram stain missed. Even if CSF cultures are negative because of antibiotic pretreatment, the Gram stain is still more sensitive than BAD testing in our setting in detecting bacterial meningitis. The Gram stain also detects infection earlier than culture and is just as fast as BAD testing.

## Conclusions

For a serious illness such as bacterial meningitis the most important quality of a test for its detection is that it misses as few cases as possible, ie it must be a sensitive test. The poor sensitivity of the bacterial antigen detection kit results in the detection of bacterial meningitis in our population does not recommend its use even though it is one of the criteria listed in the NHMRC and Centre for Disease Control, Atlanta case definitions for bacterial meningitis.

Even when BAD use is restricted to a population with greater probability of having bacterial meningitis (those with >30 cells/μL) it is still costly, and has no advantage in timing of diagnosis or sensitivity compared with the cheaper, routine Gram stain.

## Editorial

This article documents the disappointing experience of the RDH laboratory with bacterial antigen testing for bacterial meningitis. Of particular concern from a public health perspective, is the low sensitivity of the BAD test in diagnosing meningococcal disease (42.9%). BAD testing does, however, continue to be used in other laboratories, including laboratories within the NT, with varying levels of acceptability.

Comparison of the RDH results with the sensitivity of the BAD test battery during the 1987-1991 outbreak of meningococcal disease in Central Australia suggests that the BAD test can inform

There is still a three to seven day turnaround for return of serotyping specimens, which are sent from Darwin to the Prince of Wales Hospital in Sydney. There may be a public health use for this test in early serogrouping of culture proven meningitis when it is very useful to know whether it is a strain with an effective vaccine eg A or C.

Some possible explanations for the conflicting literature reports include high rates of Hib meningitis in developing countries (vaccination is thought to be responsible for the plummeting rates in our population) and the greater sensitivity of that particular latex test. Perhaps the better facilities available for staining and culture in developed countries compared with those in developing countries also contribute to the limited utility of latex tests in our context.

## Action taken

The Microbiology Laboratory has introduced a procedure whereby CSF specimens are cytocentrifuged to enhance the sensitivity and specificity of Gram stains. As a result of this survey and review of the literature the laboratory will be phasing out BAD testing over the next months.

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public health action during outbreaks of invasive meningococcal disease, especially when the turn around time for serogrouping is long.[1] The Central Australian outbreak resulted in 77 notified cases of meningococcal disease most of which were caused by serogroup A *N meningitidis,* a vaccine preventable strain. There were 43 cases confirmed on culture and/or CSF microscopy, 7 probable cases diagnosed on the basis of a positive CSF latex result and 10 cases suspected on clinical grounds that were epidemiologically linked to confirmed cases.

Overall, 75.3% of cases had a positive BAD test (58 cases). The sensitivity of the test in diagnosing definite cases of meningococcal disease in this outbreak (Table) was significantly higher than that experienced testing sporadic cases at RDH ($p<0.01$). Factors influencing the difference in performance of the test in these two situations may include the different case definitions of bacterial meningitis (>1 x $10^9$ leukocytes, >75% polymorphs was used in Central Australia), experience with the test and the spectrum of disease severity during the outbreak.[2] (Note * comment below the table for an explanation of the effect of prevalence on sensitivity and specificity).

Unfortunately, there is insufficient information from the Central Australian study to determine specificity, positive and negative predictive values against all cases of bacterial meningitis reported during the outbreak or the cost of BAD testing.

Improvements in the national laboratory surveillance of meningococcal disease, such as the National Neisseria Network, mean that serogrouping results can be obtained much more quickly than in the past, so that BAD testing may no longer be necessary even in outbreak situations.

**Table Sensitivity of bacterial antigen testing in the 1987-91 meningococcal disease outbreak in Central Australia**

| Case definition | Number "true" positive | BAD test positive | Sensitivity (%) |
|---|---|---|---|
| Definite cases | | | |
| CSF or blood culture positive | 43 | 35 | 81.4 |
| CSF microscopy positive | 17 | 16 | 94.1 |
| All definite cases | 60 | 51 | 85.0 |
| Confirmed Serogroups A or C | 33 | 27 | 81.8 |
| Confirmed Serogroup B | 4 | 3 | 75.0 |

* Although disease prevalence has more effect on predictive value rather than sensitivity and specificity, these measures can vary when the mix (mild versus severe) of patients with the disease varies when their prevalence varies.

The editors are interested to hear from other laboratories on their experience with BAD testing and its cost effectiveness.

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# Enhanced influenza surveillance in the Top End

*Sue Reid, CDC, Darwin*

On 1 February 1998, in response to recommendations put forward by the Australian Influenza Pandemic Planning Committee, a three month period of enhanced influenza surveillance commenced in the Top End. So far, enhanced surveillance activities have involved the participation of 33 sentinel general practitioners, health care providers in rural and remote communities, medical, nursing and administrative staff at Royal Darwin Hospital (RDH), Top End high school nurses, RDH laboratory, Western Diagnostic Pathology and Queensland Medical Laboratory.

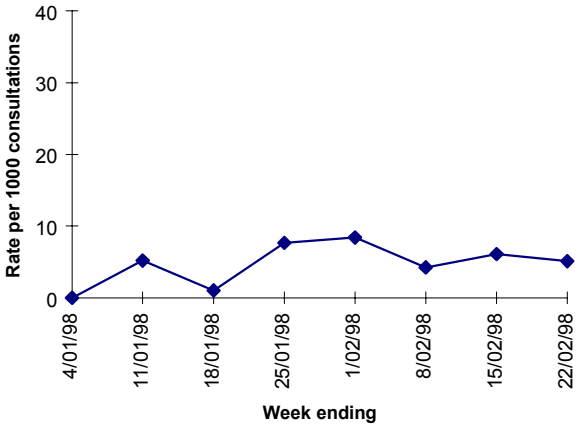
Sixty one throat washes collected from individuals who strictly met the clinical case definition of influenza were forwarded to the WHO Collaborating Centre for Influenza Reference and Research in Melbourne of which 58 have been processed. We have recently received confirmation of one case of influenza A ($H_3$). Although the strain is yet to be confirmed, preliminary tests suggest that it is the Sydney-like strain. Also, a recent serology result from an RDH inpatient proved positive for influenza B.

Weekly consultation rates for clinical influenza recorded through the Top End sentinel influenza surveillance scheme shows a constant low level of influenza activity since the beginning of the year, with rates ranging from 0-8.4 per 1000 consultations (Figure). Although recently there have been anecdotal reports of people being off sick with the 'flu', the large number of negative influenza results from WHO suggest that other common respiratory pathogens are currently circulating in the community.

Top End high school and RDH nursing staff absenteeism data are also being collected during the three month surveillance period as an indirect measure of influenza activity. CDC will be plotting changes in absenteeism over time looking for apparent trends or clustering.

A full report on the enhanced surveillance activities will be available in the next edition of the *Bulletin*.

**Figure Clinical cases of influenza per 1000 consultations, NT Top End**




# Interrupting transmission of the common cold

Acute viral respiratory infections are major health problems causing significant morbidity and subsequent social and economic burden worldwide. The control of infection for most of the associated agents has been difficult to achieve, and hence the interruption of transmission plays an important role in reducing their impact on the community.[1] However, when is a person most contagious and what can be done to minimise the risk of transmission?

Concentrations of some rhinovirus serotypes, the most frequent cause of the common cold, can be found in nasal washing's as soon as 8 hours after exposure, peaking at about 48 hours.[2] Between 24 and 72 hours after infection, nasal discharge is most voluminous and this, together with viral concentration, makes the first three days of a cold the most contagious period.[2]

Transmission can occur either directly through aerosols or indirectly by contact with contaminated surfaces (fomites), and household spread is important in maintaining a link among mixing groups such as school, workplace and neighbourhood clusters.[3] Importantly, while respiratory disease is an occupational hazard for health care workers,[4] they have in turn been identified as sources of infection in outbreaks.[5] Therefore, reducing the risk of transmission during the acute phase can be achieved through a few simple measures:

1. Good personal hygiene: regularly washing face and hands, particularly after coughing and sneezing.
2. Use tissues (appropriately disposed of after a single use) instead of handkerchiefs to stifle coughs and sneezes.
3. Stay at home when unwell, in particular avoid sending children to school if they are acutely unwell.
4. Clean surfaces contaminated by respiratory droplets after coughing and sneezing.

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# More rain and more melioidosis - the 1997/98 wet season

*Bart Currie,[1,2] Adam Jenney,[1] Gary Lum,[1] Dale Fisher[1] and Angela Merianos[3]*
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The wetter than predicted monsoon has led to a record number of confirmed cases of melioidosis. Until late March there have been 35 cases of culture confirmed melioidosis, with 4 deaths. Eight cases occurred in Katherine since the flood, with 1 death. and one case before the flood. This is the highest number of cases in Katherine since surveillance began for melioidosis in 1991. Although failing to reach statistical significance, the higher numbers are temporally associated with the topographical changes due to torrential rain and flooding. Factors in Katherine contributing to the higher incidence include the much greater exposure of Katherine residents to infected water and mud this wet season and heightened surveillance for the milder manifestations of melioidosis, both among clinicians and exposed residents. As expected the commonest presentation has been pneumonia, with skin ulcers, genitourinary infections (especially prostatic abscesses), septic arthritis and osteomyelitis also occurring. Four of the Katherine cases presented with skin ulcers. All deaths have been in patients with known risk factors. There have been 2 children with melioidosis.

The following points are of note:

1. Suspect and culture for melioidosis in any diabetic with a septic illness in the Top End, especially in the wet season.
2. Test for diabetes in all melioidosis cases, and aim for tight control of blood glucose during therapy.
3. Diagnosis is increased by using selective culture media (modified Ashdown's Broth) with frequent sampling (sputum, throat, rectal and ulcer swabs), and collection of standard blood cultures. Clinicians should liaise with laboratory staff to ensure selective media are available, including for remote communities. Remember to take standard sputum and ulcer swabs as well, as selective broth will not allow growth of most other common organisms.
4. Mortality is decreased by early diagnosis and appropriate therapy.
5. Follow up of cases and adherence by both treating doctors and patients to eradication therapy (usually at least 3 months of antibiotics after discharge) is critical to prevent relapse, which can be fatal.
6. The Top End empirical treatment protocol for adult community-acquired pneumonia is devised to cover both melioidosis in patients with risk factors, as well as other important pathogens (see table below).
7. Once melioidosis is confirmed the treatment recommended is:

   Initial INTENSIVE THERAPY for usually 14 days (longer if organ/deep tissue abscesses, septic arthritis or osteomyelitis) of:

   ⇒ intravenous high dose ceftazidime

   plus either

   ⇒ high dose cotrimoxazole OR high dose doxycycline.

   This is followed by ERADICATION THERAPY for at least 3 months of:

   ⇒ oral monotherapy with either high dose cotrimoxazole or doxycycline.
8. This wet season, selected patients have been started on continuous ceftazidime infusions via PICC lines (peripherally inserted central catheters). This enables early discharge from hospital and home supervision of the ceftazidime infusions using a pre-prepared plastic pressure infusion bottle which requires no batteries or electricity and is changed every 24 hours.

   Certain patients have been switched to meropenem instead of ceftazidime for the intensive therapy. A large trial has been underway in Thailand comparing ceftazidime with imipenem (similar to meropenem), and results will inform future antibiotic choices for melioidosis.

**Table Initial therapy of adult community-acquired pneumonia in the Top End[1]**

| | Mild pneumonia | Moderate pneumonia | Severe pneumonia |
|---|---|---|---|
| No risk factors[2] present | Penicillin | Penicillin | Ceftriaxone |
| Risk factors[2] present | Penicillin | Ceftriaxone plus gentamicin | Ceftriaxone or ceftazidime plus gentamicin |

1. For 'atypical pneumonia' consider adding erythromycin.
2. Risk factors include: alcohol, diabetes, chronic lung disease, chronic renal failure and steroid therapy.

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## NT NOTIFICATIONS OF DISEASES BY DISTRICTS 1997 AND 1996

| DISEASES | ALICE SPRINGS | | BARKLY | | DARWIN | | EAST ARNHEM | | KATHERINE | | TOTAL | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | '97 | '96 | '97 | '96 | '97 | '96 | '97 | '96 | '97 | '96 | '97 | '96 |
| Acute Rheumatic Fever | 8 | 10 | 1 | 0 | 7 | 17 | 0 | 9 | 5 | 8 | 21 | 44 |
| Adverse Vaccine Reaction. | 5 | 3 | 0 | 0 | 8 | 8 | 1 | 0 | 2 | 1 | 16 | 12 |
| Amoebiasis | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| Arbovirus infections | | | | | | | | | | | | |
| Barmah Forest Virus | 4 | 1 | 6 | 0 | 22 | 21 | 4 | 3 | 7 | 2 | 43 | 27 |
| Kunjin Virus | 0 | 0 | 2 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 2 | 1 |
| Dengue | 0 | 0 | 0 | 0 | 5 | 6 | 2 | 0 | 0 | 0 | 7 | 6 |
| Ross River Virus | 54 | 1 | 23 | 5 | 113 | 84 | 4 | 17 | 29 | 24 | 223 | 131 |
| Campylobacter | 81 | 56 | 2 | 2 | 97 | 162 | 5 | 3 | 21 | 39 | 206 | 262 |
| Chlamydia | 243 | 222 | 37 | 21 | 256 | 232 | 61 | 59 | 59 | 111 | 656 | 645 |
| Congenital Syphilis | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 1 | 2 | 1 |
| Donovanosis | 22 | 8 | 0 | 3 | 5 | 4 | 0 | 1 | 3 | 5 | 30 | 21 |
| Gastroenteritis | 0 | 0 | 0 | 0 | 3 | 2 | 0 | 0 | 0 | 7 | 3 | 9 |
| Glomerulonephritis | 6 | 4 | 1 | 0 | 7 | 6 | 1 | 4 | 0 | 2 | 15 | 16 |
| Gonococcal Disease | 425 | 317 | 50 | 14 | 358 | 210 | 81 | 81 | 125 | 163 | 1039 | 785 |
| Gonococcal Conjunct. | 100 | 0 | 6 | 0 | 0 | 0 | 1 | 1 | 0 | 1 | 107 | 2 |
| *Haemophilus inf* type b | 3 | 3 | 0 | 0 | 0 | 2 | 0 | 0 | 1 | 0 | 4 | 5 |
| Hepatitis A | 27 | 15 | 5 | 5 | 35 | 33 | 0 | 7 | 26 | 16 | 93 | 76 |
| Hepatitis B | 2 | 2 | 3 | 0 | 8 | 1 | 1 | 1 | 5 | 1 | 19 | 5 |
| Hepatitis C (incidence) | 0 | 0 | 0 | 0 | 1 | 3 | 0 | 0 | 0 | 0 | 1 | 3 |
| Hepatitis C (prevalence) | 40 | 31 | 3 | 3 | 293 | 174 | 2 | 3 | 4 | 6 | 342 | 217 |
| Hepatitis D | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| HIV infections | 1 | 1 | 0 | 0 | 9 | 6 | 0 | 0 | 1 | 0 | 11 | 7 |
| HTLV-1 | 26 | 21 | 0 | 1 | 4 | 3 | 0 | 0 | 3 | 2 | 33 | 27 |
| Legionnaires Disease | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 1 | 2 | 2 |
| Leprosy | 0 | 0 | 0 | 0 | 1 | 3 | 1 | 2 | 0 | 1 | 2 | 6 |
| Leptospirosis | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 |
| Malaria | 6 | 2 | 0 | 0 | 25 | 23 | 3 | 1 | 3 | 0 | 37 | 26 |
| Measles | 3 | 13 | 0 | 0 | 4 | 4 | 1 | 0 | 3 | 9 | 11 | 26 |
| Meningococcal Infect. | 6 | 0 | 0 | 0 | 5 | 7 | 3 | 1 | 1 | 1 | 15 | 9 |
| Mumps | 3 | 1 | 0 | 0 | 6 | 4 | 0 | 0 | 1 | 0 | 10 | 5 |
| Pertussis | 5 | 5 | 0 | 9 | 13 | 1 | 1 | 1 | 5 | 0 | 24 | 16 |
| Pneumococcal Disease | 50 | 36 | 0 | 4 | 29 | 26 | 1 | 0 | 1 | 9 | 81 | 75 |
| Rotavirus | 47 | 75 | 14 | 6 | 150 | 60 | 30 | 2 | 18 | 10 | 259 | 153 |
| Rubella | 3 | 0 | 1 | 0 | 3 | 7 | 0 | 0 | 0 | 0 | 7 | 7 |
| Salmonella | 84 | 119 | 12 | 14 | 176 | 190 | 34 | 38 | 41 | 61 | 347 | 422 |
| Shigella | 74 | 69 | 15 | 7 | 44 | 35 | 13 | 17 | 23 | 21 | 169 | 149 |
| Syphilis | 148 | 156 | 27 | 1 | 35 | 55 | 21 | 31 | 40 | 47 | 271 | 290 |
| Tuberculosis | 4 | 8 | 0 | 0 | 24 | 15 | 2 | 2 | 4 | 6 | 34 | 31 |
| Typhoid | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 2 | 0 |
| Typhus | 0 | 0 | 0 | 0 | 2 | 2 | 0 | 0 | 0 | 0 | 2 | 2 |
| Yersiniosis | 0 | 0 | 0 | 0 | 3 | 2 | 0 | 0 | 0 | 0 | 3 | 2 |
| **Total** | **1483** | **1179** | **208** | **95** | **1754** | **1410** | **274** | **284** | **432** | **556** | **4151** | **3524** |

**Points to note regarding notifications:**

- Australian Encephalitis (MVE), botulism, brucellosis, chancroid, cholera, congenital rubella syndrome, diphtheria, Hepatitis E, hydatid disease, listeriosis, lymphogranuloma venereum, poliomyelitis and viral haemorrhagic fever are all notifiable but had "0" notifications in this period.

# NOTIFICATION DATA

In this edition of the *Bulletin*, we have expanded the usual notification report to include the following tables:

- Notification of Diseases by Districts, 1997 and 1996 (number of cases);
- Notification of Diseases by Districts, 1997 and 1996. Incidence rates per 100 000;
- Table 1: Notification of Disease, Alice Springs 1997;
- Table 2: Notification of Disease, Barkly 1997;
- Table 3: Notification of Disease, Darwin 1997;
- Table 4: Notification of East Arnhem 1997;
- Table 5: Notification of Disease, Katherine 1997.

The convention nationally is to report diseases by date of report rather than date of onset. Therefore, a number of cases with illness onset in 1996 were reported in 1997 and appear in the 1997 statistics.

Tables 1-5 present Notifiable Disease data by indigenous status, sex and age group for cases with complete data in all fields, and a grand total that includes all cases. This means that for all jurisdictions, the grand total is greater than the sum of totals for each sub category. Indigenous status "Unknown" has been included in the table for Darwin because 37.6% of notifications failed to identify indigenous status.

Overall, data were complete in 98%, 94%, 59%, 97% and 98% of notifications from Alice Springs, the Barkly, Darwin, East Arnhem and Katherine respectively.

The Editorial staff have included these tables so that service providers in each district have greater access to the crude data on notifiable diseases in their area. We would be interested in comments about their utility.

## Comments on number of cases

*Acute rheumatic fever*
Numbers of acute rheumatic fever in the Top End are inconclusive for 1997 due to the fact that CDC is not being notified of all new diagnoses, and the existing rheumatic heart disease database has not been updated since March 1997.

*Campylobacteriosis*
There was a large difference in the age distribution among cases of campylobacteriosis in the Darwin District between 1996 and 1997. 59% of cases were aged under 2 years in 1996 while only 36% were under 2 years in 1997 ($p=0.056$, NS). Most of the cases occurred in three rural communities in January to April 1996.

*Dengue*
Dengue virus is not endemic in the NT. Most of the 1996 and 1997 cases were acquired in Indonesia.

*Donovanosis*
Alice Springs had a special, dedicated donovanosis project worker who was active in case identification, notification and follow-up.

*Gonococcal disease*
The increase in gonococcal figures are due to a combination of increased screening and probably tampon testing.

Refer to the *NT Comm Dis Bull* Vol 4 No 3 Sept 1997 for details of the 1997 outbreak of non-sexually transmitted gonococcal conjunctivitis.

*Malaria*
Of the 37 positive cases of malaria in 1997, 11 used appropriate prophylaxis, 26 either used no prophylaxis or inappropriate treatment.

*Meningococcal infection*
There was an increase in the number of cases of meningococcal meningitis cases in 1997. All were sporadic cases of disease.

*Pertussis*
The NT did not experience the pertussis outbreak in 1997 that affected most States in Australia.

*Rotavirus*
All districts except Alice Springs experienced increases in rotavirus activity in 1997, especially Darwin and East Arnhem. Outbreaks usually occur from June to August in the Top End, while Alice Springs experienced increased rotavirus activity in January through May in 1996.

*Syphilis*
There has been a sustained decrease in the number of reported syphilis cases since 1994, except for the Barkly where increased screening and improved reporting started in 1997

## NOTIFICATION OF DISEASES BY DISTRICTS, 1997 AND 1996
## INCIDENCE RATES PER 100,000

| DISEASES | ALICE SPRINGS | | BARKLY | | DARWIN | | EAST ARNHEM | | KATHERINE | | TOTAL | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | '97 | '96 | '97 | '96 | '97 | '96 | '97 | '96 | '97 | '96 | '97 | '96 |
| Acute Rheumatic Fever | 22.0 | 27.5 | 15.0 | 0.0 | 6.7 | 16.2 | 0.0 | 67.4 | 31.1 | 49.7 | 11.8 | 24.8 |
| Adverse Vaccine Reaction | 13.8 | 8.3 | 0.0 | 0.0 | 7.6 | 7.6 | 7.5 | 0.0 | 12.4 | 6.2 | 9.0 | 6.8 |
| Amoebiasis | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 6.2 | 0.0 | 0.6 |
| Arbovirus infections | | | | | | | | | | | | |
| Barmah Forest Virus | 11.0 | 2.8 | 90.0 | 0.0 | 20.9 | 20.0 | 29.9 | 22.5 | 43.5 | 12.4 | 24.2 | 15.2 |
| Kunjin Virus | 0.0 | 0.0 | 30.0 | 0.0 | 0.0 | 1.0 | 0.0 | 0.0 | 0.0 | 0.0 | 1.1 | 0.6 |
| Dengue | 0.0 | 0.0 | 0.0 | 0.0 | 4.8 | 5.7 | 15.0 | 0.0 | 0.0 | 0.0 | 3.9 | 3.4 |
| Ross River Virus | 148.6 | 2.8 | 344.9 | 75.0 | 107.4 | 79.8 | 29.9 | 127.3 | 180.2 | 149.1 | 125.5 | 73.7 |
| Campylobacter | 222.8 | 154.1 | 30.0 | 30.0 | 92.2 | 153.9 | 37.4 | 22.5 | 130.5 | 242.3 | 115.9 | 147.4 |
| Chlamydia | 668.5 | 610.7 | 554.8 | 314.9 | 243.2 | 220.4 | 456.7 | 441.7 | 366.6 | 689.7 | 369.1 | 362.9 |
| Congenital Syphilis | 0.0 | 0.0 | 0.0 | 0.0 | 1.9 | 0.0 | 0.0 | 0.0 | 0.0 | 6.2 | 1.1 | 0.6 |
| Donovanosis | 60.5 | 22.0 | 0.0 | 45.0 | 4.8 | 3.8 | 0.0 | 7.5 | 18.6 | 31.1 | 16.9 | 11.8 |
| Gastroenteritis | 0.0 | 0.0 | 0.0 | 0.0 | 2.9 | 1.9 | 0.0 | 0.0 | 0.0 | 43.5 | 1.7 | 5.1 |
| Glomerulonephritis | 16.5 | 11.0 | 15.0 | 0.0 | 6.7 | 5.7 | 7.5 | 29.9 | 0.0 | 12.4 | 8.4 | 9.0 |
| Gonococcal Disease | 1169.2 | 872.1 | 749.7 | 209.9 | 340.1 | 199.5 | 606.4 | 606.4 | 776.7 | 1012.8 | 584.6 | 441.7 |
| Gonococcal Conjunctivitis | 275.1 | 0.0 | 90.0 | 0.0 | 0.0 | 0.0 | 7.5 | 7.5 | 0.0 | 6.2 | 60.2 | 1.1 |
| *Haemophilus inf* type b | 8.3 | 8.3 | 0.0 | 0.0 | 0.0 | 1.9 | 0.0 | 0.0 | 6.2 | 0.0 | 2.3 | 2.8 |
| Hepatitis A | 74.3 | 41.3 | 75.0 | 75.0 | 33.3 | 31.4 | 0.0 | 52.4 | 161.6 | 99.4 | 52.3 | 42.8 |
| Hepatitis B | 5.5 | 5.5 | 45.0 | 0.0 | 7.6 | 1.0 | 7.5 | 7.5 | 31.1 | 6.2 | 10.7 | 2.8 |
| Hepatitis C (incidence) | 0.0 | 0.0 | 0.0 | 0.0 | 1.0 | 2.9 | 0.0 | 0.0 | 0.0 | 0.0 | 0.6 | 1.7 |
| Hepatitis C (prevalence) | 110.0 | 85.3 | 45.0 | 45.0 | 278.4 | 165.3 | 15.0 | 22.5 | 24.9 | 37.3 | 192.4 | 122.1 |
| Hepatitis D | 2.8 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.6 | 0.0 |
| HIV infections | 2.8 | 2.8 | 0.0 | 0.0 | 8.6 | 5.7 | 0.0 | 0.0 | 6.2 | 0.0 | 6.2 | 3.9 |
| HTLV-1 | 71.5 | 57.8 | 0.0 | 15.0 | 3.8 | 2.9 | 0.0 | 0.0 | 18.6 | 12.4 | 18.6 | 15.2 |
| Legionnaires Disease | 2.8 | 0.0 | 0.0 | 0.0 | 0.0 | 1.0 | 7.5 | 0.0 | 0.0 | 6.2 | 1.1 | 1.1 |
| Leprosy | 0.0 | 0.0 | 0.0 | 0.0 | 1.0 | 2.9 | 7.5 | 15.0 | 0.0 | 6.2 | 1.1 | 3.4 |
| Leptospirosis | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 6.2 | 0.0 | 0.6 | 0.0 |
| Malaria | 16.5 | 5.5 | 0.0 | 0.0 | 23.8 | 21.9 | 22.5 | 7.5 | 18.6 | 0.0 | 20.8 | 14.6 |
| Measles | 8.3 | 35.8 | 0.0 | 0.0 | 3.8 | 3.8 | 7.5 | 0.0 | 18.6 | 55.9 | 6.2 | 14.6 |
| Meningococcal Infections | 16.5 | 0.0 | 0.0 | 0.0 | 4.8 | 6.7 | 22.5 | 7.5 | 6.2 | 6.2 | 8.4 | 5.1 |
| Mumps | 8.3 | 2.8 | 0.0 | 0.0 | 5.7 | 3.8 | 0.0 | 0.0 | 6.2 | 0.0 | 5.6 | 2.8 |
| Pertussis | 13.8 | 13.8 | 0.0 | 135.0 | 12.4 | 1.0 | 7.5 | 7.5 | 31.1 | 0.0 | 13.5 | 9.0 |
| Pneumococcal Disease | 137.5 | 99.0 | 0.0 | 60.0 | 27.6 | 24.7 | 7.5 | 0.0 | 6.2 | 55.9 | 45.6 | 42.2 |
| Rotavirus | 129.3 | 206.3 | 209.9 | 90.0 | 142.5 | 57.0 | 224.6 | 15.0 | 111.8 | 62.1 | 145.7 | 86.1 |
| Rubella | 8.3 | 0.0 | 15.0 | 0.0 | 2.9 | 6.7 | 0.0 | 0.0 | 0.0 | 0.0 | 3.9 | 3.9 |
| Salmonella | 231.1 | 327.4 | 179.9 | 209.9 | 167.2 | 180.5 | 254.5 | 284.5 | 254.8 | 379.0 | 195.2 | 237.4 |
| Shigella | 203.6 | 189.8 | 224.9 | 105.0 | 41.8 | 33.3 | 97.3 | 127.3 | 142.9 | 130.5 | 95.1 | 83.8 |
| Syphilis | 407.1 | 429.1 | 404.9 | 15.0 | 33.3 | 52.3 | 157.2 | 232.1 | 248.5 | 292.0 | 152.5 | 163.2 |
| Tuberculosis | 11.0 | 22.0 | 0.0 | 0.0 | 22.8 | 14.3 | 15.0 | 15.0 | 24.9 | 37.3 | 19.1 | 17.4 |
| Typhoid | 2.8 | 0.0 | 0.0 | 0.0 | 1.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 1.1 | 0.0 |
| Typhus | 0.0 | 0.0 | 0.0 | 0.0 | 1.9 | 1.9 | 0.0 | 0.0 | 0.0 | 0.0 | 1.1 | 1.1 |
| Yersiniosis | 0.0 | 0.0 | 0.0 | 0.0 | 2.9 | 1.9 | 0.0 | 0.0 | 0.0 | 0.0 | 1.7 | 1.1 |
| **Total** | **4079.7** | **3243.4** | **3118.9** | **1424.5** | **1666.4** | **1339.6** | **2051.4** | **2126.2** | **2684.2** | **3454.7** | **2335.6** | **1982.8** |

**Points to note regarding interpretation of rates:**

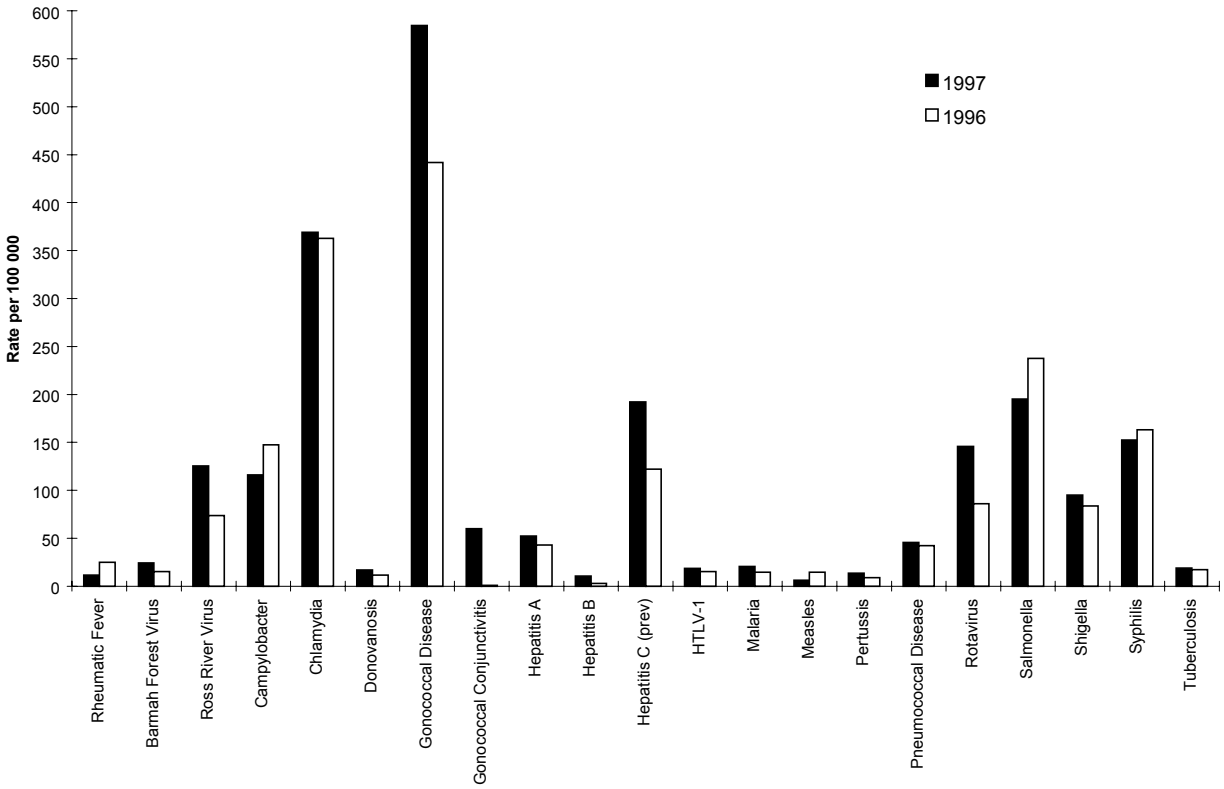
- Note: these rates are based on the year of reporting, not the year of illness onset. This is national convention in the reporting of Notifiable Diseases.
- Rates should be interpreted in the context of population size. Each case has greater weight as population size decreases.
- One case in Alice Springs, the Barkly, East Arnhem and Katherine respectively, is equivalent to 2.9, 15.8, 7.9 and 6.5 cases in Darwin.

## NOTIFIED CASES OF VACCINE PREVENTABLE DISEASES IN THE NT BY REPORT DATE 1997 AND 1996

| DISEASES | TOTAL | | No. cases among children aged 0-5 years | |
|---|---|---|---|---|
| | '97 | '96 | '97 | '96 |
| Congenital rubella syndrome | 0 | 0 | 0 | 0 |
| Diphtheria | 0 | 0 | 0 | 0 |
| *Haemophilus influenzae* type b | 4 | 5 | 4 | 3 |
| Hepatitis B | 19 | 5 | 2 | 0 |
| Measles | 11 | 26 | 6 | 14 |
| Mumps | 10 | 5 | 4 | 1 |
| Pertussis | 24 | 16 | 7 | 3 |
| Poliomyelitis, paralytic | 0 | 0 | 0 | 0 |
| Rubella | 7 | 7 | 2 | 4 |
| Tetanus | 0 | 0 | 0 | 0 |

- Mumps is largely under-reported.

## NT WIDE NOTIFIABLE DISEASES 1997 AND 1996




Rates <10/100 000 not listed
NT est. resid. pop - 177,730 as supplied by Epidemiology & Statistical Branch, THS

Excel sheet (1)

Excel sheet (2)

Excel sheet (3)

## NT MALARIA NOTIFICATIONS
## October to December 1997

*Compiled by Merv Fairley, CDC, Darwin*

Five notifications of malaria were received for the third quarter of 1997. The following table provides details about where the infection was thought to be acquired, the infecting agent and whether chemoprophylaxis was used.

| ORIGIN OF INFECTION | REASON EXPOSED | AGENT | CHEMO-PROPHY-LAXIS | COMMENTS |
|---|---|---|---|---|
| PNG | Resident | *P.vivax* | No | Diagnosed RDH. Past exposure. |
| INDONESIA | Holiday | *P.vivax* | No | Diagnosed RDH. Past exposure. |
| S.E.ASIA | Holiday | *P.vivax* | No | Diagnosed Alice Springs. |
| INDONESIA | Holiday | *P.vivax* | Yes | Diagnosed RDH. Prophylaxis compliance poor. |
| INDONESIA | Fisherman | *P.vivax* | No | Diagnosed RDH. Bought medication from local Indonesian shop and took it only when felt unwell. |

# CORRECTION

## Update: NT retrospective search for lyssavirus in humans

The previous version of this table appeared in error in the December issue of the *Bulletin* (Vol 4, No 4, page 20). The correct version appears below. Please note that the number of cases diagnosed with viral meningitis was 66.

**Table 2 Diagnosis on review of cases found using eight ICD-9 codes for potential encephalitis, RDH, January 1992-September 1996**

| Review diagnosis | No. of cases | % |
|---|---|---|
| ***Congenital/Chronic*** | | |
| Lead encephalopathy secondary to petrol sniffing* | 37 | 24.0 |
| Cerebral palsy unknown cause* | 3 | 1.9 |
| Congenital HSV infection* | 1 | 0.6 |
| Familial leucoencephalopathy* | 1 | 0.6 |
| ***Non-infective*** | | |
| Steroid encephalopathy | 1 | 0.6 |
| Cerebrovascular accident | 1 | 0.6 |
| Unstable epilepsy* | 1 | 0.6 |
| Fractured base of skull with secondary encephalitis | 1 | 0.6 |
| ***Infectious*** | | |
| Viral meningitis* | 66 | 42.8 |
| Unexplained illness with headache and fever* | 8 | 5.2 |
| Murray Valley encephalitis | 8 | 5.2 |
| Pneumococcal meningoencephalitis/brain abscess | 2 | 1.3 |
| Partially treated bacterial meningitis* | 2 | 1.3 |
| Herpes encephalitis | 1 | 0.6 |
| Cryptococcal meningitis | 1 | 0.6 |
| Rotavirus encephalitis | 1 | 0.6 |
| Meningococcal meningitis* | 1 | 0.6 |
| ***Unexplained encephalitis*** | 18 | 1.7 |
| Total | 154 | 100.0 |

* Not considered to have encephalitis

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# STAFF UPDATES

## ALICE SPRINGS

**Virginia Sitzler** left Disease Control, Alice Springs on 6 March and ventured forth to Melbourne to study interior decorating for a year. **Jenny Hain's** (formerly **Rossiter)** leave to fill the position of Infection Control Coordinator at Alice Springs Hospital has been extended for another year. **Belinda Farmer**, who used to work in Disease Control, Katherine and most recently at Community Care, Alice Springs is now acting in the Public Health Nurse position.

The Master of Applied Epidemiology (MAE) students have commenced their studies at the National Centre for Epidemiology and Population Health (NCEPH) at ANU, Canberra and will arrive in Alice Springs in April to commence their field placement in the Population Health Unit. **Dan Ewald** is a medical graduate and completed a Masters in Public Health and Tropical Medicine at James Cook University last year. He has previously worked in Alice Springs and Central Australia in a number of settings and has been working for the last few years in the Torres Strait. **Christine Franks** has an ethnography background and has worked in Central Australia for many years, most recently as an HIV/STD educator within the Population Health Unit. She has been selected into the newly established MAE (Indigenous Health) stream.

## DARWIN

**Vicki Krause** left for Oxford, UK on 11 March and will be on sabbatical/recreation leave until September this year. **Angela Merianos** will be acting Director of Disease Control, as well as Head of Surveillance for the six months. The part-time Head of Immunisation position is expected to be filled soon.

**Joan Fong**, who has worked in the area of leprosy control since the early 1970s will commence long service leave from 1 April. Her position will be filled by **Lesley Scott** who worked in Disease Control in Katherine from 1986 to 1992 and then at Timber Creek as a remote area nurse for 5 years.

**Terry O'Brien** left CDC at the end of February to take up a Clinical Nurse Consultant TB position in Brisbane.

**Sarah Huffam** (former Infectious Diseases Registrar) was recently appointed into the STD Registrar position in Clinic 34.

**Jacki Mein** (former STD Registrar, AIDS/STD Unit and Microbiology Registrar, RDH) will return from the first residential block at NCEPH, ANU, Canberra and commence her MAE field placement in Disease Control at the beginning of April. The Top End MAE Indigenous Health place has been filled by **Halijah Mokak** who will be based at the Epidemiology Branch in Health House. Prior to being selected for the MAE program, Halijah was working as a physiotherapist at Carpentaria Disability Services.

## NHULUNBUY

**Hartley Dentith,** who has worked in Disease Control in Nhulunbuy for many years has recently been appointed Coordinator of the Unit. **Liz Stephenson** (formerly AIDS/STD Unit, Darwin) will commence in the AIDS/STD Public Health Nurse position on 14 April and **Nilva Egana**, who has been relieving for three months is due to finish on 17 April. **Erin Stewart** recently left the Unit to return to a part-time position in Gove hospital.

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